CHRONIC DERMATOPHYTOSIS – A CLINICOMYCOLOGICAL STUDY

DISSERTATION

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CERTIFICATE

This is to certify that this dissertation entitled **"CHRONIC DERMATOPHYTOSIS – A CLINICOMYCOLOGICAL STUDY"** is a bonafide work done by Dr. S. KARTHIKA, Postgraduate student of Department of Dermatology and Leprosy and Institute of STD, Madras Medical College, Chennai – 600 003 during the academic year 2003 – 2006 for the award of degree of M.D. (Dermatology, Venereology and Leprosy) – Branch XII A. This work has not previously formed the basis for the award of any Degree or Diploma.

Prof.B.Parveen, M.D.D.D.,
Professor and H.O.D.
Department of Dermatology & Leprosy,
Madras Medical College,
Chennai – 600 003.

Prof.Dr.Kalavathy Ponniraivan, B.Sc., M.D.,
DEAN
Madras Medical College &
Govt. General Hospital
Chennai – 600 003.

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CONTENTS

INTRODUCTION

Dermatophytosis is a superficial fungal infection of keratinized tissue caused by dermatophytes. They are a group of taxonomically related fungi that utilize keratin as a source of nutrients and colonise keratinized tissues including stratum corneum of epidermis, nail, hair, and horny tissue of animals.[1]

Though a superficial infection, dermatophytes do evoke inflammatory responses like scaling, vesiculation, pustulation, and sometimes abscess formation, because of their metabolic activities. Thus clinical infection occurs if fungi penetrate the host's protective barrier.

Dermatophytosis is the leading cause of cutaneous fungal infection and accounts for those of the cutaneous fungal infection related medical expenses. Chronic dermatophytosis is a social, economical, psychological burden not only to the patient, but also to those closely associated with them. They act as source of infection constantly spreading with or without their knowledge. Remissions and exacerbations mark the course of the disease and

chronic dermatophytosis still remains a challenge to the practicing dermatologist.

Multiple factors may affect the incidence of this fungal infection within a population. These include geographic area, climate, immunocompetence of the host, pathogenicity of the agent and availability of the treatment.[2] The growing number of immunocompromised patients due to chemotherapy, transplant, HIV, etc has led to increased incidence of dermatophytosis.

This study aims to bring out the recent trends in the age, sex, distribution, causative species, factors associated with in chronic dermatophytosis of and more than three years duration.

AIM OF STUDY

The aim of this study is to assess the following in cases of chronic dermatophytosis:

- Age, sex distribution.
- Clinical types of dermatophytosis.
- Characteristic features of lesions or morphological features.
- Probable factors responsible for chronicity.
- Various isolates encountered
- Blood group association.

MATERIALS AND METHODS

This study was conducted in the Mycology section of Department of Dermatology, Madras Medical College and Government General Hospital, Chennai – 3 from December 2003 to December 2005.

Patient having dermatophytosis for a duration of 3 years and above were selected for the study after obtaining informed consent.

Detailed case history of each patient with reference to the occupation, symptoms, duration of infection, treatment taken, contact with animals or other infected persons were recorded.

Clinical features like sites of involvement, morphology of lesions, percentage of body surface area affected, other associated systemic and cutaneous disorders were noted. The size of one palm was taken as 1%.

The morphology of lesions with reference to presence of inflammation, definition of margin, pigmentary changes and presence of central clearance were recorded. The pattern of nail involvement was also noted.

Blood grouping and typing was done for all the patients. Blood haemogram, blood sugar, and urea and absolute Eosinophil count were also done to detect associated disorders. Blood VDRL for syphilis and ELISA for HIV infection were done in high risk patients.

Mycological examination.

Skin and nail scrapings were collected from all the patients and examined in 10 – 40%. Potassium hydroxide mount for presence of fungal elements.

All the above specimens were subjected to culture. In the case of skin lesions, the site was cleaned with 70% alcohol before collecting the specimen. Inoculation was done on modified Sabouraud's Dextrose Agar with and without cycloheximide and chloramphenicol. All the inoculated slants were incubated at 26^{o} C and observed for growth of colonies for a period of 4 weeks.

The isolates were studied for the rate of growth, macroscopic morphology and pigment production. Subcultures in Cornmeal agar were done to differentiate *Trichophyton rubrum* from *Trichophyton mentagrophytes* according to the persistent pigmentation. Microscopic examination for the morphological

characters of the isolates were done in Lactophenol cotton Blue mount.

All the patients were treated with topical 2% clotrimazole cream and systemic griseofulvin in dose of 10mg/kg body weight for a duration of 4 – 6 weeks. Those who did not respond to the above were started on systemic fluconazole 150mg biweekly or ketoconazole 200 mg od for 4 to 6 weeks. One patient with nail infection was treated with oral terbinafine 250mg od for 3 months.

REVIEW OF LITERATURE

HISTORY:

In 1910, Raymond Sabouraud (1864 – 1938), the father of modern mycology classified dermatophytosis according to site of involvement in his treatise '*Les Tiegnes'*. In 1934, Emmon's view of dermatophytic taxonomy resulted in the three genera known today: *Trichophyton*, *Microsporon, Epidermophyton*.[3]

Chronic dermatophytosis was defined by Kamaan(1978) as a refractory infection which persisted for more than one year with or without recurrence or a total duration of more than one year[4]. Hay & Brostoft (1982) considered it as a persistant infection for 3 years in spite of treatment with **griseofulvin** for 3 months.[5]

Racial and genetic factors:

Tinea imbricata is endemic in Far East, West Pacific, and parts of Central and South America[6]. *Trichophyton violaceum* infection is common in Jews, *Microsporon ferrugineum* in North China, Korea, and Japan. Strain variation can affect the infectivity of particular group of individuals. Differences have been noted in

the strains, infecting the aboriginals and non aboriginals in Australia.

No HLA has been associated with chronic dermatophytosis[7]. An Autosomal Dominantly inherited genetic factor for susceptibility may be responsible for family clusters and racial preferences of chronic anthropophilic dermatophytic infection especially. *Trichophyton rubrum*. This factor causes differences in keratin which may affect the ability of the fungus to attack and digest the protein.[8,9,10]

Chronic dermatophytosis is common in North Africa, where there is high rate of consanguineous marriage. This suggests Autosomal Recessive inheritance of genetic anamoly possibly associated with deficient cellular immunity[11]. Similar pattern of inheritance has been noted for genetic susceptibility factor in Tinea imbricata[6].

PATHOMECHANISM

Characteristically, dermatophytes restrict themselves to dead keratinized tissues except in rare cases where patients are immunosuppressed[12]. The amount of keratin is most important for the affinity of the fungus[13].

Pathological changes occur within 4 hours of infection. At which, maximum adherence of microconidia to keratinocytes occur. Within 3 days of infection, epidermal thickness increases mainly due to edema[14,15].

Chronically infected animals showed changes similar to those at peak of primary infection. In addition, there is infiltration of dermis by mast cells. There is an increase in the number of epidermal cell layers. [14].

Studies of chronic extensive dermatophytosis are made in athymic nude mice(BALB/c or BALB/k) using different strains of *Trichophyton mentagrophytes* and *Trichophyton quinckaneum*[14].

AGENT FACTORS

In some cases of chronic dermatophytosis, a mycological cell wall glycoprotein was found to cross react with the human A isoantigen. Because of this ,immunological tolerance fungal proliferation goes unchecked[16].

Mannan produced by *Trichophyton rubrum* suppresses or diminishes the inflammatory responses[17].

The fungus secretes keratinases that allow the dermatophtyes to burrow deeper into the stratum corneum[18]. These enzymes also act as virulence factors[19].

HOST DEFENCE

Once the fungal hyphae invade the viable tissue, their cell wall activate the alternative complement pathway resulting in neutrophil chemotaxis[18,20]. These cells adhere to the opsonised fungal hyphae and kill them through myeloperoxidase (MPO – H2O2 –cl) pathway. Thus they prevent invasion and sepsis by the dermatophytes even in absence of specific immunity. Cytotoxins produced by this mechanism were found to be fungicidal for *Trichophyton rubrum* [21].

The inflammatory reaction provoked by these interaction can increase the epidermal turnover time and thereby force retreat of the fungus back into stratum corneum or eliminate them through desquamation[23]. Nonspecific mechanism of defence prevent invasion into dermis and blood stream, for e.g., serum inhibitory factor depletes iron, an essential nutrient for the fungus[20].

IMMUNITY

Cell mediated immunity is the corner stone of host defence and instrumental in eradication of the fungus. It provides protection from re-infection after primary dermatophytosis[22,18] Absence of CMI predisposes to chronic infection with increased severity[20]. Patients with chronic dermatophytosis have relatively specific defect in delayed hypersensitivity to trichophytin. This

may be due to selective allergy to the antigen or due to dermatophyte derived lymphocyte inhibitory factor as suggested by earlier studies[23].

HLA DR ,a Class II MHC Ag is the surface marker for activated T cells. In an in vitro study, this was observed on inhibited lymphocytes cocultured with Ag, suggesting that the primary target for the inhibitory effect in vitro is lymphocyte, rather than the Antigen presenting cell. In this in vitro study, no evidence for circulating dermatophyte derived lymphocyte inhibitory factor was found in sera of chronic dermatophytosis patients. Further, a direct inhibitory effect of *Trichophyton rubrum* on lymphocyte proliferation to recall Ag was observed. This was reversible and not associated with loss of cell viability[23].

In a study of chronic dermatophytosis unresponsive to griseofulvin, 58% showed immediate hypersensitivity to trichophytin as well as other fungal and nonfungal allergens, while 11% showed delayed hypersensitivity response[24]. In another study 26% showed immediate reactions[25].

Immediate wheal reactions to intradermal trichophytin antigen is frequent in chronic *Trichophyton rubrum* infection and

this has been noted in atopics with or without tinea infection. This does not mean sensitization to dermatophytes but is primarily a sign of cross reactivity to molds. It can be passively transferred with serum, which demonstrates the presence of specific IgE antibody in these patients[128]. In a study specific IgE antibody towards. *Trichophyton rubrum* was demonstrated in patients with chronic infection. But it was absent in patients with tinea capitis[26].

IgE plays a role in CMI suppression perhaps through histamine activity[26]. Patients who display IgE mediated immediate hypersensitivity reaction to trichophytin test instead of DTH response are prone to chronic dermatophytosis usually by *Trichophyton rubrum*[27]. T - lymphocyte response to trichophytin was reduced in *Trichophyton rubrum* infection in contrast to *Trichophyton mentagrophytes* in a study[28]. Thus in persistent dermatophytosis, hypersensitivity to dermatophyte antigen possibly play an important role in pathogenicity[26]

Trichophyton rubrum is suited to survive on skin surface and it uses different strategies. Infected patients cannot elicit CMI to eliminate the fungus but may initiate IgE mediated immediate type of reaction. Trichophytin tests are negative at 48 hrs despite

chronic persistent and widespread infection[29]. *Trichophyton rubrum* makes more mannan that usually suppress CMI. Mannan act by inhibiting critical steps in Ag processing or presentation thus inhibiting immune reaction induction[30,18]. *Trichophyton rubrum* is not aggressive when compared with other species. By remaining in the stratum corneum, it may evade immune surveillance, complement, and polymorphs. Spores can survive off human body and remain plentiful in human habitats[29].

In chronic infections, inflammation is minimal because of suppressed DTH response by host. Differences in Ag penetration through skin may prevent induction of immunity. Perhaps neonatal exposure to fungus or cross reactivity to moulds may induce tolerance by confusing Ag recognition of self vs nonself. Persistant infection induces immune unresponsiveness by activating specific suppressors T cells. These cells suppress proliferation of the T. rubrum specific T. cell[27].

AGE AND SEX INCIDENCE

Dermatophytosis commonly occurs at 11 – 20 yrs of age. But chronic dermatophytosis was observed to affect males at 20 – 40 yrs and females at 30 – 40 yrs[30]. In another study males were commonly affected in 3rd decade; whereas, it was common between 4th and 5th decade in females. The mean age incidence was 39 yrs[118]. Males were affected in younger age than females[31].

Higher incidence of infection was observed in males except in certain occasional instances where females out numbered males.

Onychomycosis is common in elderly and males are 2.99 times more affected[32,33,34]. *Trichophyton rubrum* infection is 3 times more common in males[35,36,37], especially in 10 – 20 years age group[38]. *Trichophyton raubitschekii* also has male preponderance [39].

Etiological Agents Causing Chronic Dermatophytosis

Trichophyton rubrum

It is the most common isolate in most studies [40,30,41,42]. The isolation rates of *Trichophyton rubrum* in various studies were

45.74% [43]
55.18% [44]
80% [45]
93% [24]
87% [31]

It is the common cause of chronic infections especially when associated with Icthyosis[43], Psoriasis[46]. It is also found to be common in renal transplant patients [47] and griseofulvin unresponsive patients[24]. It is the commonest organism causing onychomycosis in pre-exisiting tinea pedis[10,45,4] especially in coal miners 45, non autoimmune patients.

In HIV infection, it causes proximal subungual onychomycosis and foot abcesses due to deeper invasion[48]. This occurs as a result of altered function, chemistry and metabolism of its arthospores[49].

In diabetics, recurrent *Trichophyton rubrum* infection manifests as one palm – two sole syndrome [50].

It causes widespread and multiple site infection in renal transplant patients. [51,47,4]

Trichophyton rubrum is not aggressive as compared with other species. Its ability to evade host defences and remain as spores in human habitats accounts for its ubiquitous presence and high prevalence[29]. The lack of cutaneous inflammation at site of infection may reflect its local inhibitory effect.[23]

Colony[41]

Downy form

Most commonly isolated form in chronic infection. The colony is white downy or cottony and domed. Reverse is initially dark brown and later deep red.

Melanoid form – Similar to downy form. Brown pigment diffuses into the medium and masks the red pigment on the reverse.

Dysgonic form – slow growing, brittle, unstable, deep red colonies, reverts to downy form

Granular form – cream to pink, powdery or granular colonies with raised and folded surface. Reverse is red brown.

Yellow form – downy colonies with yellow reverse

Microscopy [41].

- Tear drop shaped microconidia in enthryse distribution is characteristic in downy colonies. Rarely engreppe distribution is also seen.

- Pencil shaped macroconidia are seen in granular colonies

Special test [41] :

- Urease test is negative

- In vitro hair perforation test is negative

Trichophyton raubitschekii[39]

It is a variant of *Trichophyton rubrum* causing Tinea corporis and Tinea manuum in Asian and African countries

Colony : Similar to *Trichophyton rubrum* with brown reverse

Microscopy : *Trichophyton rubrum* like macroconidia

Trichophyton mentagrophytes

It is the second most common species[41]. It was isolated in 26.6% of patients in one study [43]. Another study showed 71% isolation rate of 29%[52]

It is the common cause of tinea pedis, tinea capitis, tinea corporis, tinea cruris and tinea barbae[53]. Frequently it has been isolated in patients with renal transplantation[54], long standing Diabetes mellitus and hereditary palmoplantar keratoderma[13].

It has been reported to cause extensive and deep dermatophytosis and folliculitis[95] in a HIV infected patient with history of intravenous drug abuse and CD_4 count 335 cells/mm^3 [55].

Partial cross reactivity between *Trichophyton rubrum* and *Trichophyton mentagophytes* have been noted. It is a potent sensitizer than *Trichophyton rubrum* ($P < 0.01$)[56]. Host specific strains have emerged which will probably separate as species like in *Trichophyton mentogrophytes var interdigitale*[41].

Colony [41]

Anthropophilc species forms white fluffy downy colonies while zoophilic form flat, granular colonies with aerial hyphae.

Reverse is pale yellow. Sometimes the reverse is red to yellow brown in colour, indistinguishable from *Trichophyton rubrum*. Persistant yellow colour is produced in Cornmeal agar.

Microscopy [41]

Numerous spherical microconidia in enthryse or engreppe distribution along with cylindrical macroconidia and spiral hyphae are seen.

Special tests :

- Urease test is positive
- Perforating organs are seen in hair in vitro

Trichophyton tonsurans

It has taken over *Trichophyton rubrum* as the common isolate[57] causing tinea capitis, tinea corporis, tinea cruris and tinea pedis. In most cases of tinea corporis gladiatorum the isolates have been *Trichophyton tonsurans*[58,59]. It can cause inflammatory tinea capitis mimicking bacterial infections in adults[60]. It is seen especially in black children in US and Canada probably because of their hairstyle and hygiene habits[41]. It has been reported as a cause of for an outbreak of nosocomial infection in an old age nursing home[61].

Colony [41]:

Colonies have wrinkles and folds with short aerial hyphae giving a seude appearance. The reverse is rich red to brown in colour.

Microscopy [41]

Balloon like microconidia in enthryse and engreppe distribution along with cigar like macroconidia are characteristic.

Special tests [41]

- Urease positive – growth stimulated by thiamine
- In vitro hair perforation test is variable

Trichophyton violaceum

Earlier studies showed it to be the commonest isolate[14]. It commonly causes tinea capitis. An unusual presentation of encapsulated abscess has been reported to have been caused by it in a patient with defective CMI. This defect along with malnutrition, hypoadrenalism and griseofulvin resistance gave unusual lesions and chronicity in above said patient [62].

Colony : [41]

Colonies are waxy, leathery, deep violet and slow growing. Reverse is violet in colour.

Microscopy [41]:

Macroconidia are absent and microconidia are rare, hyphae are distorted and have chlamydoconidia.

Special test [41]: Growth and sporulation stimulated by thiamine

Trichophyton verrucosum

It is a significant cause of human dermatophytosis in rural areas. It causes tinea barbae, tinea capitis, tinea corporis and occasionally onychomycosis[53]. Usually localized inflammatory lesion with pustules are seen. But extensive non pustular, non inflammatory tinea corporis has been reported in HIV patients[63].

Colony [41]:

Slow growing dull white or gray to yellowish tan coloured waxy colonies with colourless reverse.

Microscopy [41]: Chains of chlamydoconidia are characteristic. Macroconidia are absent.

Special test [41] – Require thiamine and inositol for growth.

Trichophyton simii[41]

It is the frequent cause of ring worm in monkeys and chicken in endemic areas. It has been regularly isolated from soil and small mammals. Occasionally human beings become victims of infection. Interfamilial infections with this agent was noted in a study of tinea capitis. In South Indian families (Kamalam) though highly infectious, the infrequent incidence of infection was considered to be due to probable alteration of the fungus while passing through the human host, losing its virulence[31].

Colony : Flat granular buff coloured colonies with a central umbo are seen. Reverse is yellow to vinaceous (red brown).

Microscopy : Thin walled, smooth and clavate cylinderiform or fusiform macroconidia are seen. The macroconidial cells may enlarge and become thick walled to form endochlamydoconidia. Clavate, elongated pyriform microconidia are produced laterally on hyphae.

Special test

- Urease test is positive
- Hair perforation test is negative

Epidermophyton floccosum

It is the third most common agent causing tinea corporis, tinea cruris and onychomycosis. It was the first agent to be identified as causative of tinea pedis [53].

Colony [41]

Colonies are wrinkled or folded and olive green or khaki in colour with fine fuzzy texture. Reverse is buff coloured.

Microscopy [41]:

Clusters of multiseptate club shaped smooth, thick walled macroconidia are characteristic. Microconida are absent.

Microsporon gypseum :

It is a geophilic organism isolated from soil, worldwide causing ectothrix infection of hair [53], Disseminated dermatophytosis caused by Microsporon gypseum has been reported in advanced AIDS [64]. Epidemics have occurred with this agent. It causes occupational dermatosis among gardeners [31].

Colony : Fast growing colonies are flat, light tan to medium brown or cinnemon brown, powder or velvety in texture. Reverse is reddish brown in colour.

Microscopy : Numerous spindle shaped rough walled macroconidia with 4-6 septa and flagelliform appendix at tip is characteristic. The microconidia are pyriform.

Transmission of Infection

Modes of disease transmission include person to person contact, handling of contaminated laundry and use of shared razor[65]. Fomites are significant in transmission of tinea infection. However host factors like occupation immunological status, local factors like trauma due to increased physical activity, increased humidity due to occlusive synthetic clothing, poor personal hygiene due to low socioeconomic status also play a role[53].

Occlusive foot wear provide high humidity, which is conducive for fungal growth. High incidence of tinea unguium is seen due to trauma of nails as a result of hard physical work and barefoot walking [48,66].

Familial infections were reported in a number of studies mostly with anthropophilic dermatophytes. *Trichophyton rubrum* was the common agent to be reported in a case of conjugal infection[31].

Large quantities of spores in non inflammatory type of tinea capitis was mostly responsible for intrafamilial infections in a study [31].

Traditional and religious habits like cohabitation and ritual ablution may affect the prevalence in certain population[67].

Clinical Presentation

Tinea corporis is the commonest presentation[30,43]. Waist is the commonest site in chronic and non chronic infection. While groin and back are common in non chronic and chronic disease respectively[4]. High humidity and temperature were related to the higher incidence[27] especially in coal miners[68,69] and wrestlers[58]. Patient with advanced HIV disease have more severe and diffuse skin involvement[48] which is more resistant to therapy. Secondary lichenification can occur in chronic *Trichophyton rubrum* infection of thighs and feet [70].

Tinea pedis is most common site of infection in psoriasis chronic patients resistant to griseofulvin[24], atopy[36] and hereditary palmoplantar keratoderma. In a study of diabetes with mycotic infection, 82% had tinea pedis and there was increased risk of diabetic foot syndrome. Moreover cracks and fissures in tinea pedis may get secondarily infected in diabetes [38].

Tinea pedis was observed in 64% of HIV patients. The entire plantar aspect may become infected and event spread to dorsum of foot in severe immunocompromised states [48].

Tinea cruris is more common in males[71.] About 70% of dermatophytosis were in the groin region[66]. In recurrent or persistant tinea cruris, besides potential carriage in clinically normal sites, hypersensitivity to the antigen play an important role in pathogenecity[72]

Tinea faciei when chronic, resembles discoid lupus erythematosus[73]. It can be misdiagnosed as photosensitivity when bilaterally symmetrical[74,73].

Tinea unguium is the most common nail disorder[75] in adults especially elderly[71]. Males are commonly affected[34]. Diabetics are

more prone for distal superficial onychomycosis type[76]. Proximal subungual white onychomycosis is common in renal transplant patient, [119] and HIV diseaese[48] (88.7%). Damage to nail by invading fungus is mechanical rather than chemical action[77]. Infected nails contribute to widespread and multiple site infection in chronicity with *Trichophyton rubrum*[4].

The various predisposing factor for tinea unguium include immunosuppression, poor peripheral circulation warm moist environment, increasing age, nail trauma, tinea pedis and family history[78,45,79]. Diabetes mellitus, hypercholesterolaemia, cardiovascular disease, osteoarthritis may also predispose[78]. About 1/3rd of diabetes mellitus patients are affected especially those with peripheral vascular disease[34]. In them the thick brittle nail may cause injury to surrounding skin leading to secondary infection and complication. Recurrence in some are due to early termination of treatment[80], poor penetration of antifungals to the centre of infection[81], family history, occupation, life style or underlying physiology and concomintant disease[80].

Tinea imbricata caused by *Trichophyton concentricum* is a chronic endemic infection acquired in childhood. Multiple

concentric rings covering atleast half of the skin surface is characteristic and the scales are abundant with spores[5].

Atypical presentation in chronicity

Deep dermatophytosis have been reported in immunocompromised patients due to long term steroids and chemotherapy. Most of them are due to *Trichophyton rubrum*. They present as subcutaneous abscesses, papules, nodules and

Majocchi's granuloma over unusual sites like thighs, buttocks, inguinal folds and face[82]. Tinea faciei mimicking seborrheic dermatosis, palmar infection similar to keratoderma blenorrhagica, erythroderma like widespread, tinea corporis have been reported in HIV and Cushings disease[31].

Factors associated with chronicity

Atopy :Chronic dermatophytosis is 3 fold more frequent in atopic dermatitis. The incidence of atopy was 49 out of 106 patients in a study [30]. Atopy was considered as a major and important factor in predisposition and persistence of dermatophytosis. Environmental factors and atopy were associated factors in 81% and 77% respectively [83]. In a study, there was relative risk of 3.1 for contracting tinea pedis when being an atopic or having family

history of atopy[36]. In a study of atopic patients, 50% with tinea and 40% without tinea showed immunological hypersensitivity to trichophytin and increased IgE levels[84]. Atopic dermatitis can be exacerbated by chronic dermatophytic infection and trichophytin hypersensitivity. The resultant recalcitrant eczema resolves following systemic antifungal therapy. This has been known as 'Atopic-chronic dermatophytosis syndrome' by Jones (1973)[85].

Icthyosis:

Icthyosis vulgaris was the most common cutaneous association in a study [30]. *Trichophyton rubrum* was found to cause palmo plantar hyperkeratosis and nail infection in a case of Icthyosis vulgaris which was resistant to topical steroids and emollients. The coexistence of dermatophytosis and icthyosis were more frequent than visualized, but the difference in diagnosis was considered to be due to typical manifestation with the presence of icthyosis. Dermatophytosis is common in X-linked icthyosis and icthyosis vulgaris because of slow turn over of epidermis and delayed desquamation of stratum corneum respectively [31].

Psoriasis:

In a study with 34 psoriasis patients having dermatophytosis, 20 had tinea pedis, 6 had tinea cruris, and 2 had tinea manuum. *Trichophyton rubrum* was the isolate in all these patients. Lesions were intermingled with psoriatic plaques. These findings suggest that possibility of fungal manifestation in psoriasis would not appear to be an exceptional occurence [46]. In another study prevalence of pedal onychomycosis was 13% in psoriasis, with 56% more chances than non psoriasis patients in acquiring the nail infection [86].

OTHERS:

Chronic dermatophytosis is also frequently associated with collagen vascular diseases [10], acne, cutaneous tags, contact dermatitis with kumkum, hypertrichosis, seborrheic warts[31].

Recalcitrant tinea capitis that resemble seborrheic dermatitis can be associated with Langerhan's cell histiocytosis [87].

SYSTEMIC DISORDERS:

BRONCHIAL ASTHMA

In bronchial asthmatic patients, allergic symptoms improved dramatically with oral antifungals and relapsed of antifungal therapy after discontinuation of antifungal therapy. Etiological role for Trichophytin allergens in asthma is provided by bronchial reactivity to Trichophytin[88]. The amino acid sequence identity of Trichophytin allergens with diverse enzyme families support the dual role for these proteins in fungal pathogenesis and allergic disease. In addition to bronchial hypersensitivity, patients can have persistent eosinophilia and chronic rhinosinusitis. Absorption of fungal Ag gives rise to IgE Ab, sensitization of airways, rhinosinusitis, and symptomatic asthma mostly of late onset intrinsic type[89,88].

CUSHING'S DISEASE

In Cushing's disease, decreased resistance to infection is due to decreased CMI [75] and this allows for severe generalized spread of infection [90]

IgE mediated sensitivity to Trichophytin in patients with chronic dermatophytosis secondary to Cushing's syndrome has been demonstrated [75].

DIABETES MELLITUS:

Diabetes mellitus was found to be one of the major factors (44%) for chronicity in a study [31]. Higher incidence has been reported in earlier studies by Jolly and Carpenter (1969) (Rotham 1955). In diabetes mellitus there is no increased prevalence of dermatophytosis [91,40,30]. No correlation have been found between dermatophytosis and duration of diabetes mellitus, complications and blood sugar level[76]. Onychomycosis is common in diabetes mellitus. Neglected acute and chronic tinea pedis can become a problem for them [33]. The dry moccasin type tinea pedis is often underestimated by diabetics as dry skin [38]. Recurrent *Trichophyton rubrum* infection are common in diabetes mellitus [92].

RENAL TRANSPLANT:

In a study of renal transplant patients site specific or species specific difference was less marked when compared with control [54]. Non inflammatory scaly lesions without central clearing has been noted in renal transplants[47]. It has been observed that in renal transplant patients who tan early are more susceptible to fungal infections than those who burned on sun exposure. This may reflect racial susceptibility to fungal infection that is exaggerated by systemic immunosuppression[51].

STEROIDS :

Long term use of systemic steroids is associated with chronic dermatophytosis[9]. When dealing with a recalcitrant dermatosis, a dermatophyte infection has to be thought of. Diagnosis can be complicated by previous use of steroids[93]. Clinical history resembling photosensitivity and lack of clinicohistopathological correlation can also cause confusion [19].

HIV INFECTION :

In HIV the severity of the dermatophytosis is increased but not its prevalence[48]. The dermatophytosis is more varied and severe in advanced HIV – 1 infection[94]. Immunosuppression due to HIV may lead to chronic, non inflammatory extensive disease caused by zoophilic species[48,95]. Onychomycosis is common when CD4 count is approximately 370 cells/mm^3 [64] and its incidence becomes less when CD4 count falls below 200, presumably due to treatment with antifungal for other infection[48]. Tinea pedis and onychomycosis are more common in homosexual males and[108] intravenous abusers [96]. Proximal subungual onychomycosis predominates when the CD4 count declines[95]. Deep dermatophytosis[55] and Kaposi's sarcoma like lesions caused by *Trichophyton rubrum* have been reported in AIDS patients[94].

DRUG RESISTANCE AND TREATMENT FAILURE[2].

Treatment failure due to drug resistance especially griseofulvin may lead to recurrent and chronic infections. Very high MIC values have been noted in chronic infections treated with griseofulvin for long duration. The resistance may be clinical or invitro resistance [97].

Clinical resistance may be due low circulating drug level, non compliance with therapeutic regimes[81] poor penetration of drug especially in nail infection or immunosupression .Invitro resistance with high MIC levels may be due to primary innate resistance to the pharmacological agents or secondarily acquired one. Recently increased MIC levels have been observed also for ketaconazole, itraconazole in *Trichophyton rubrum* and *Trichophyton mentagrophyte* infection. The relative resistance to griseofulvin has contributed to the predominance of *Trichophyton tonsuran*s[3].

OTHERS :

Chronic dermatophytosis has also been associated with chronic mucocutaneous candidiasis, cancer chemotherapy radiotherapy and internal malignancies[31]

INVESTIGATIONS

Microscopic examination of potassium hydroxide mount

In potassium hydroxide (10-30%) mount the fungal hyphae appear as hyaline septate branching structure of even diameter. The spores are arranged in chains (arthrospores). Enhanced visualization can be achieved by adding counterstains such as Chlorazol black E, Parker's blue – black ink and Calcoflour white. The latter has to be viewed under fluorescence microscope[70].

Culture :

- Sabouraud's Dextrose Agar (SDA) is the most commonly used isolation medium. Emmon's modified SDA contains 2% Dextrose, 1% Peptone, 2-4% Agar, 0.05 gm/L chloramphenicol, and 0.5gm/L cycloheximide with pH 6.8 – 7 at temperature of 26^{o}C. This is used as selective media. Cycloheximide and chloramphenicol suppress the growth of common saprophytic and bacterial contamination respectively[98].
- **Dermatophyte Test Medium** (DTM) is useful to isolate and distinguish dermatophytes from fungal and bacterial contaminants. The colour change from yellow to red indicates growth of dermatophytes. Release of alkaline metabolites raises the pH and changes the colour of phenol red in the medium. But there are more chances of false positive and false negative results[98].

- **Dermatophyte Identification Medium** (DIM) is a simple rapid and specific method for presumptive identification of dermatophytes. If there is growth of dermatophytes, the colour of medium changes from greenish blue to purple within 24 – 48 hours after growth. It shows less false positive results with fewer fungi than DTM[98].

- **Differential medias** can be used for identification of species using nutritional requirement. For eg Thiamine for *Trichophyton violaceum*, histidine for *Trichophyton megninii* etc.[70]

- **Specialized media** such as cornmeal agar, potato flake or rice grains may be necessary to stimulate sporulation.[70]

The cultures are incubated at room temperature for upto 4 weeks before discarded as no growth. Colony characters like colour, texture, topography, rate of growth and reverse of colony for pigmentation and noted.

Microscopic examination for morphological characteristics like size, shape, topography, arrangement of spores, hyphae modification and their appendages are done in lactophenol cotton blue mount.

Physiological tests for species identification :

Urease test – This test is done on Christensen's medium for distinction between Trichophyton. rubrum and *Trichophyton mentagrophytes*. Urease produced by *Trichophyton mentagrophytes* splits urea into ammonia which raises the pH. This changes the colour of media from amber to pinkish red due to phenol red indicator. *Trichophyton rubrum* is urease negative while *Trichophyton raubitscheki*, a variant of *Trichophyton rubrum* is urease positive.[98]

In vitro hair perforation test – The test is taken as positive when the dermatophytes patient show wedge shaped perforations in the hair. It is positive in *Trichophyton mentagrophytes* and *Microsporam canis* and negative in *Trichophyton rubrum* and *Microsporan equinum*[98].

Trichophyton agars 1 – 7[70]

They are differential media for identification of species using nutritional requirement.

T1 - Casein basal medium
T2 - Inositol
T3 - Thiamine and Inositol
T4 - Thiamine
T5 - Nicotinic acid
T6 - Ammonium nitrate basal medium
T7 - Histidine

Others

Absolute eosinophil count is done as persistent eosinophilia was found in patients sensitive to dermatophyte antigen[89,60,12].

IgE levels are raised in chronically infected patients and this has been noted even in non atopics, suggestive of an immune imbalance[99,100]

PCR restriction fragment length polymorphism (RFLP) is an extremely sensitive and specific method for rapid diagnosis of dermatophytosis in which results can be obtained within 48 hours. But it cannot differentiate species. [101].

Less frequently used techniques to diagnose onychomycosis include immunohistochemistry, dual-flow cytometry and confocal microscopy[70].

Immunological test :

Intra dermal trichophytin test is done using 0.1ml of antigen derived from *Trichophyton mentagrophytes*[102] and positive results is denoted by erythema and induration of more than 10mm. If positive within 20 min, it is called immediate reaction and it is seen in chronic infection especially with *Trichophyton rubrum*. If positive after 48 hours, it is a delayed reaction and is seen in acute infection.

Histopathology

On staining with PAS or methenamine silver nitrate fungal hyphae and spores in the horny layer are seen sandwiched between two zones of cornified cells, the upper being orthokeratotic and lower parakeratotic. The presence of neutrophils in stratum corneum in another valuable diagnostic clue.

Depending on the degree of reaction of skin to presence of the fungi, one sees histological features of acute, subacute or chronic spongiotic dermatitis.

Nail biopsy taken by punch technique or scalpel under local anaesthesia will often reveal the fungus in scraping or culture negative Tinea unguium[103].

In chronic infection, large number of mast cells are seen in the dermis with more Langerhan cells in epidermis[14].

Abcesses and granulomas along with hyperkeratosis and acanthosis has been noted in chronic dermatophytosis due to genetically inherited Cell mediated immunity deficiency[11] and immunocompromised state[104].

Histological changes consistent with granuloma faciale has been noted in a Tinea faciei caused by *Trichophyton rubrum*[105].

Blood Group Association

World wide the most common blood group is 'O' (47%) followed by 'A' (41%)[106]

Incidence of dermatophytosis is high in 'O' and 'A' group but chronicity is high in blood group 'A'[107]. Studies in rabbits immunized to dermatophytosis showed that the antidermatophyte rabbit sera produced strong reaction to blood group 'A' substance and Group A erythrocyte[108].

Trichophyton rubrum is associated with blood group A but there is no evidence of increased susceptibility to dermatophytosis[108].

Blood group analysis in a group of 20 patients having *Trichophyton rubrum* infections for more than 5 years duration showed 'A' in 8 patients. B in 3 patients and 0 in 9 patients (46%)[109]. . However ABO blood group in general population in that particular area of study also appeared to be similar disputing the association of Blood group 'A' with chronicity.[110]

Blood group O was found to be more commonly associated with chronic dermatophytosis in one study.[31]

OBSERVATIONS

About 60 patients with chronic dermatophytosis for a duration of 3 years and above were chosen for this study. Of them, 24 were males and 36 were females. Females outnumbered males in this study. The male : females ratio was 1 : 1.5.

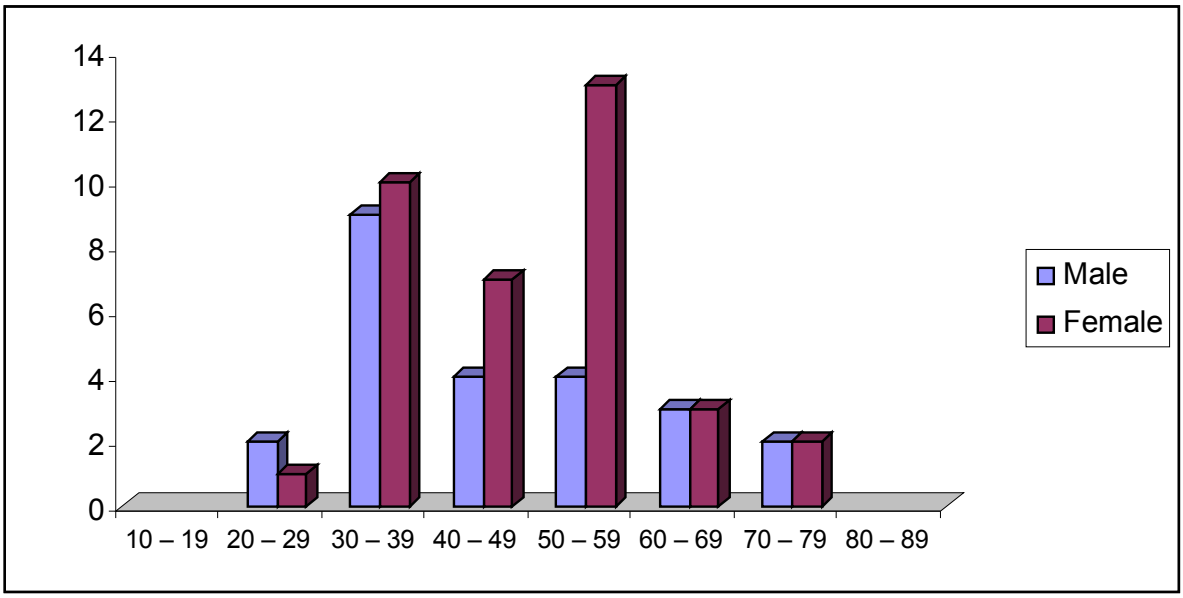
The age of the patients ranged from 23 to 75 years with a mean age of 47.18 years. The age group most commonly affected was between 30 – 40 years (Fig.1) with 10 females and 9 males in this group. Males were more in this group (Table – 1), while females were affected more between 50 – 60 years. About 13 women were in this age group.

The duration of the dermatophytosis infection varied from 3 to 35 years with a mean duration of 7.35 years.

TABLE – 1 : DISTRIBUTION OF AGE IN MALES

Age group (Years)	Male	Female
10 – 19	-	-
20 – 29	2	1
30 – 39	9	10
40 – 49	4	7
50 – 59	4	13
60 – 69	3	3
70 – 79	2	2
80 – 89	-	-

FIGURE – 1 : FREQUENCY DISTRIBUTION OF AGE



Diabetes mellitus was the most frequent systemic association observed in this study of chronic dermatophytosis. About 18 (30%) patients were diabetic(Fig.2,3). Next common association was bronchial asthma with 10 (16.6%) suffering from it. Among the asthmatics, 1 had diabetes mellitus and 2 had hypothyroidism, 4 patients were on long term steroid treatment(Fig.4,5).

2 patients of Diabetes had hypothyroidism and 1 had pemphigus vulgaris (Fig.6).

It was also observed that 2 patients had systemic lupus erythematosis, 1 had chronic obstructive lung disease, 1 had renal transplantation and 1 was hypertensive and 4 had HIV infection (Fig.7).

Cutaneous disorders like palmoplantar psoriasis (1) pemphigus vulgaris on systemic steroids. (1), candidiasis (1), discoid lupus erythematosis (2), keratolysis puncatate (1), angioedema and icthyosis vulgaris (1) were also observed in this study (Table.2).

About 2 patients gave history of irregularity in taking the antifungal drugs resulting in treatment failure.

FIGURE 2: TINEA CORPORIS IN A DIABETES MELLITUS PATIENT

FIGURE 3: TINEA AXILLARIS

FIGURE 4: TINEA CORPORIS IN A BRONCHIAL ASTHMA PATIENT ON STEROIDS

FIGURE 5 : TINEA FACIEI IN A BRONCHIAL ASTHMA PATIENT

FIGURE 6: TINEA CORPORIS IN A PEMPHIGUS VULGARIS PATIENT ON STEROIDS

FIGURE 7: TINEA CORPORIS IN A HIV PATIENT

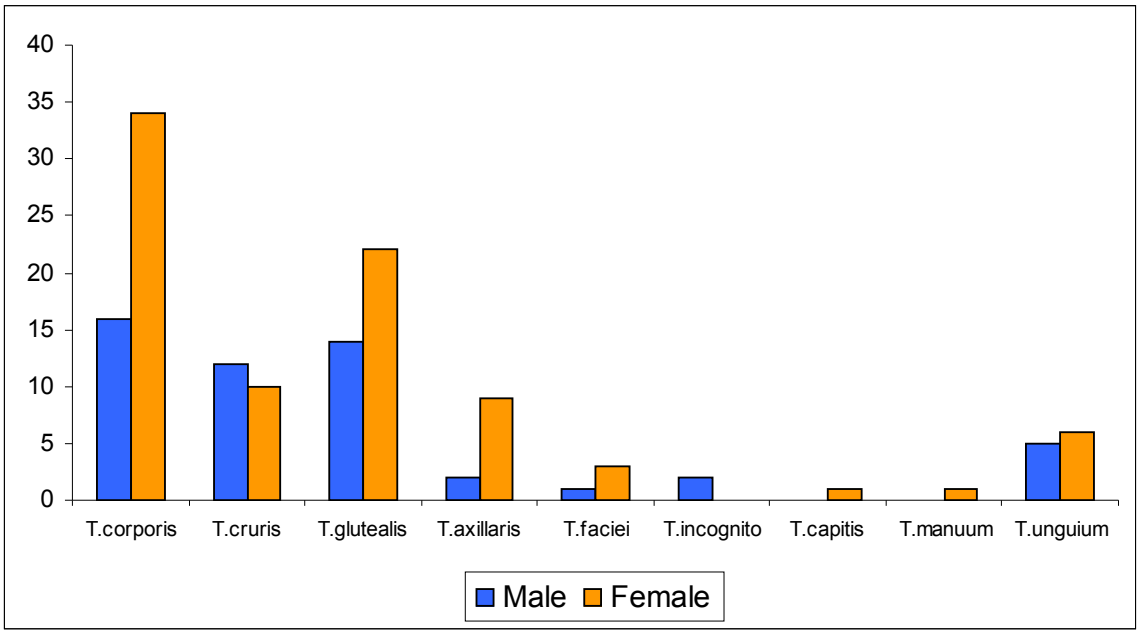
TABLE – 2 ASSOCIATED DISORDERS OBSERVED IN CHRONIC DERMATOPHYTOSIS

S. No.	Systemic Disorders	No. of patients	Cutaneous disorders	No. of patients
1.	Diabetes mellitus	16	Icthyosis vulgaris	1
2.	Bronchial asthma	8	Discoid lupus erythematosus	2
3.	Bronchial asthma on systemic steroids	4	Pemphigus vulgaris on systemic steroids.	1
4.	Human immunodeficiency virus infection	4	Keratolysis punctata	1
5.	Bronchial asthma with hypothyroidism	2	Oral candidiasis	1
6.	Treatment failure	2	Palmoplantar psoriasis	1
7.	Systemic lupus erythematosis	2	Angioedema	1
8.	Diabetes mellitus with hypothyroidism	2		
9.	Chronic obstructive lung disease	1		
10.	Renal transplantation	1		
11.	Hypertension	1		

TABLE - 3
DISTRIBUTION OF THE CLINICAL TYPES OF DERMATOPHYTOSIS

S.No.	Clinical type	Male	Female
1.	Tinea corporis	16	34
2.	Tinea glutealis	14	22
3.	Tinea cruris	12	10
4.	Tinea axillaries	2	9
5.	Tinea unguium	5	6
6.	Tinea incognito	2	-
7.	Tinea faciei	1	3
8.	Tinea manuum	-	1
9.	Tinea capitis	-	1

Fig-8 FREQUENCY DISTRIBUTION OF THE VARIOUS CLINICAL TYPES OF DERMATOPHYTOSIS



Tinea corporis was the commonest type of presentation with 94.4% of females and 66.6% of males being affected (Fig.8). The most affected site was waist in females and back in males. Most of the lesions showed hyperpigmentation and mild scaling without central clearance.

Tinea glutealis was the second commonest type in both sexes (Fig.9). 61% of females and 58% of males were affected. Tinea cruris was common in males (50%) when compared with females (27%). Similarly tinea unguium was frequent in males (20%) than females (16%)(Fig.10). But tinea axillaris was more common in females than males (Table – 3).

Multiple site involvement with more than one type of clinical presentation was seen in 78% of the patients. About 5 out of 11 cases of tinea unguium were associated with multiple site infection. In one HIV patient, proximal subungual white onychomycosis (Fig.11). was observed. All the other tinea unguium cases of distal and lateral subungual onychomycosis type.

The body surface area involved ranged from 9% to 90%. One patient had erythroderma with diffuse non inflammatory scaling and erythema involving more than 90% of body surface area.

FIGURE 9: TINEA GLUTEALIS

FIGURE 10: TINEA UNGUIUM – DISTAL AND LATERAL SUBUNGUAL TYPE

FIGURE 11: PROXIMAL SUBUNGUAL WHITE ONYCHOMYCOSIS

FIGURE 12: *T. VERRUCOSUM* INFECTION IN A HIV PATIENT

He also had Bronchial asthma and was on long term systemic steroids. The mean surface area involved was 28.3%.

Out of the 60 patients, 10 patients were lost for follow-up. Culture was done only for 50 patients and all of them had shown positive results in potassium hydroxide mount examination (Fig.13,14).

The culture positivity for dermatophyte isolation in this study was 52% (Table.4). The most frequent isolate was *Trichophyton rubrum* (Fig.16,17). It was the causative agent in about 26 (46%) patients. Out of them 3 had diabetes mellitus, 2 had Bronchial Asthma and 1 had distal subungual onychomycosis. *Trichophyton rubrum* was the isolate in the patient with erythroderma like extensive dermatophytosis.

The next frequent isolate was *Trichophyton mentagrophytes* (Fig.18,19). It was isolated in 9(34.6%) patients. Of them 3 had bronchial asthma with history of long term systemic steroids. 2 had diabetes mellitus.

The other isolates were *Trichophyton tonsurans* (2) (Fig.20,21), *Trichophyton violaceum* (1)(Fig.22,23), *Trichophyton simii* (1) (Fig.24,25) *Trichophyton* v*errucosum* (1) (Fig26,27).

Trichophyton v*errucosum* was isolated from a HIV infected patient from rural area with history of contact with cattle. He had non-inflammatory type of extensive dermatophytosis (Fig.12).

TABLE -4
AGENTS ISOLATED IN CHRONIC DERMATOPHYTOSIS PATIENTS.

S.No.	Agent	No. of Patients
1.	*Trichophyton rubrum*	26
2.	*Trichophyton mentagophytes*	9
3.	*Trichophyton tonsurans*	2
4.	*Trichophyton violaceum*	1
5.	*Trichophyton verrucosum*	1
6.	*Trichophyton simii*	1

Maximum isolates of *Trichophyton rubrum* were obtained in age group of 40 – 50 years. *Trichophyton mentagrophytes* was observed to be common in the females. The maximum number of agent isolation were done in the 30 – 50 years age groups.

About 34 (56.6%) patients were observed to have eosinophilia with their absolute eosinophil count more than 450 cells / mm^3. Among them 11 patients had diabetes mellitus and 6 patients had bronchial asthma.

MICROSCOPIC EXAMINATION OF KOH MOUNT

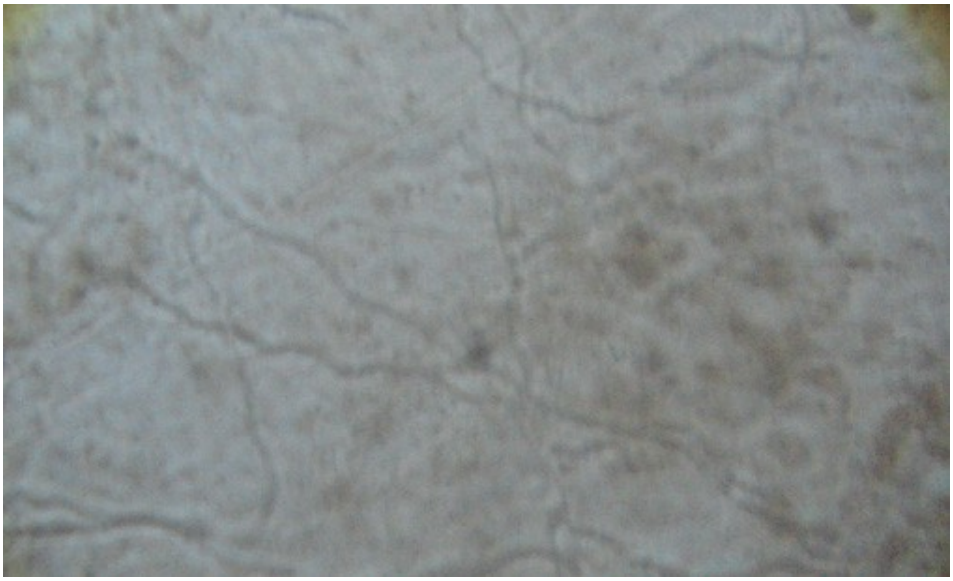


FIGURE 13: LOW POWER VIEW

FIGURE 14: HIGH POWER VIEW - ARTHROSPORES

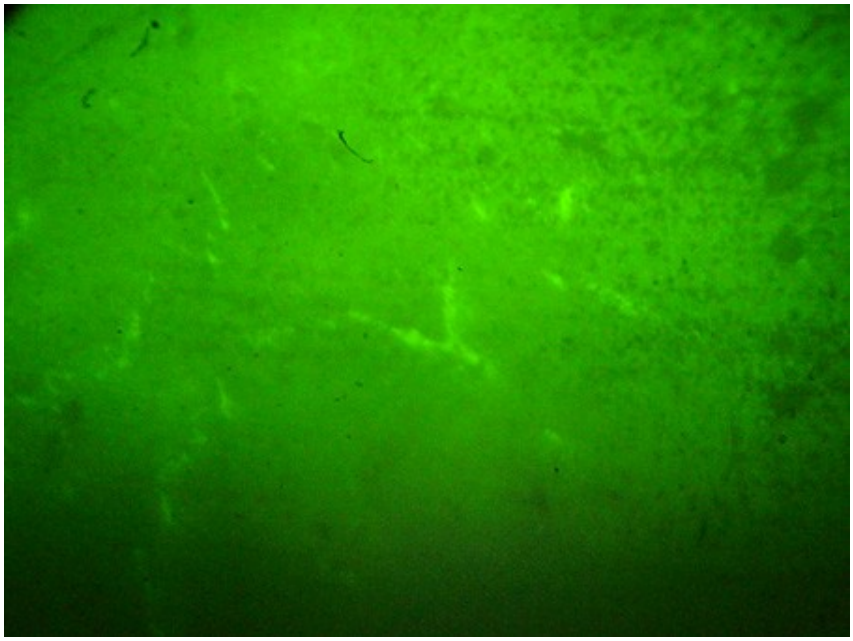


FIGURE 15: CALCOFLOUR STAINED DERMATOPHYTE HYPHAE

TRICHYPHYTON RUBRUM

FIGURE 16: COTTONY WHITE COLONY WITH RED REVERSE

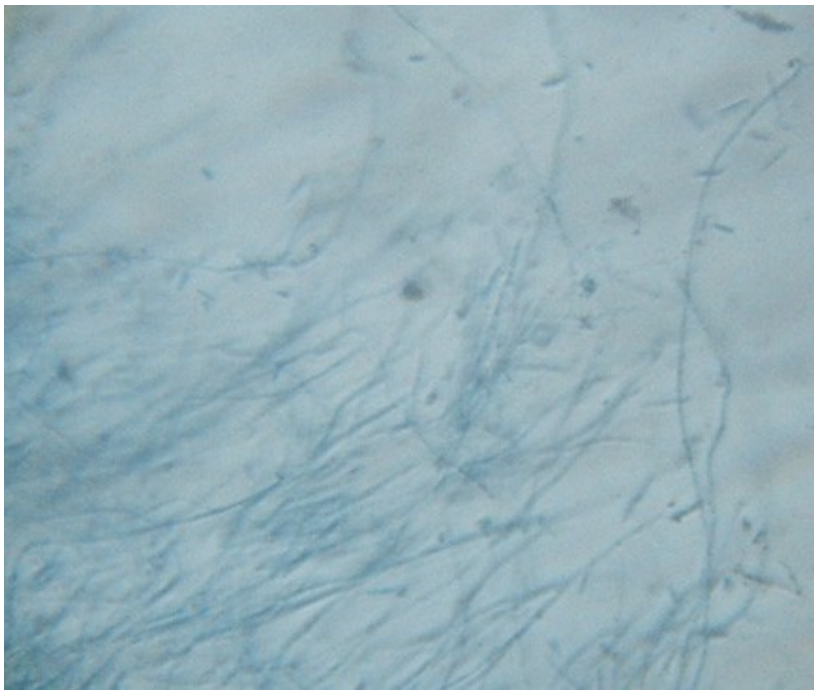


FIGURE 17 : MICROSCOPIC MORPHOLOGY – MICROCONIDIA IN ENTHRYSE DISTRIBUTION

TRICHYPHYTON MENTAGROPHYTES

FIGURE 18 : FLUFFY DOWNY COLONY WITH YELLOW REVERSE

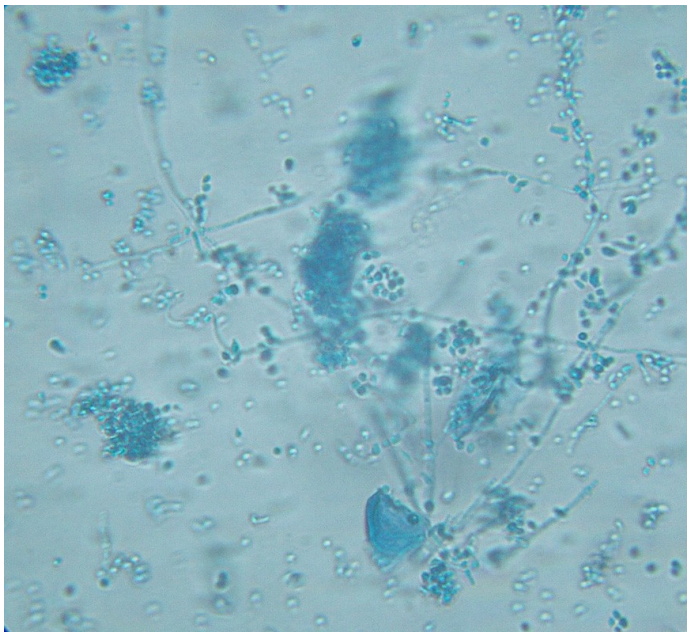


FIGURE 19 : MICROSCOPIC MORPHOLOGY – MICROCONIDIA IN ENTHRYSE AND ENGREPPE DISTRIBUTION

TRICHYPHYTON TONSURANS

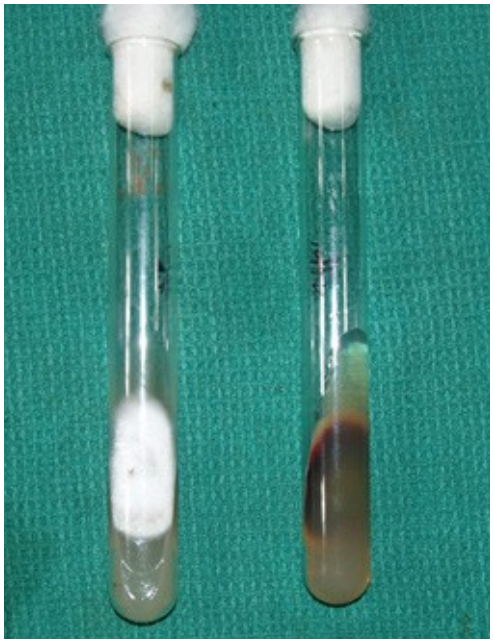


FIGURE 20 : WRINKLED COLONY WITH BROWN REVERSE

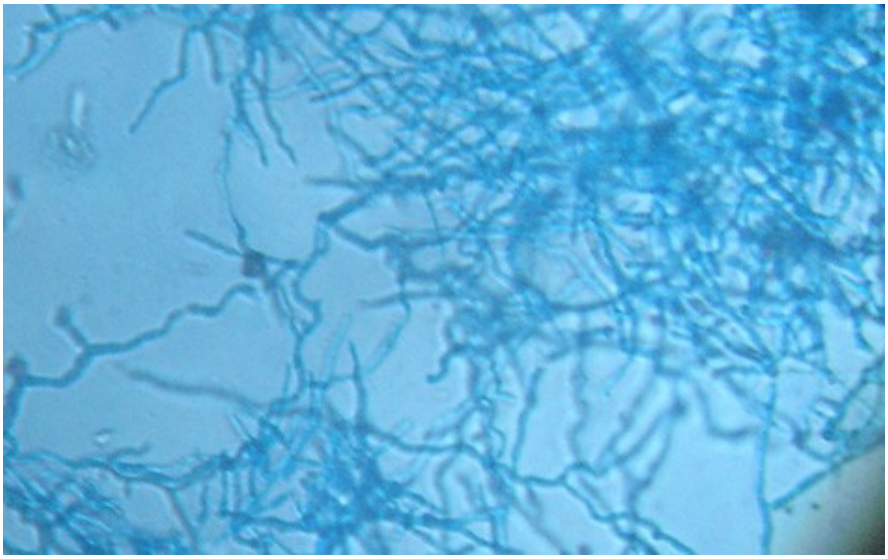


FIGURE 21 : MICROSCOPIC MORPHOLOGY – BALLOON LIKE MICROCONIDIA AND SPIRAL HYPHAE

TRICHYPHYTON VIOLACEUM

FIGURE 22 : VIOLET COLOURED COLONIES

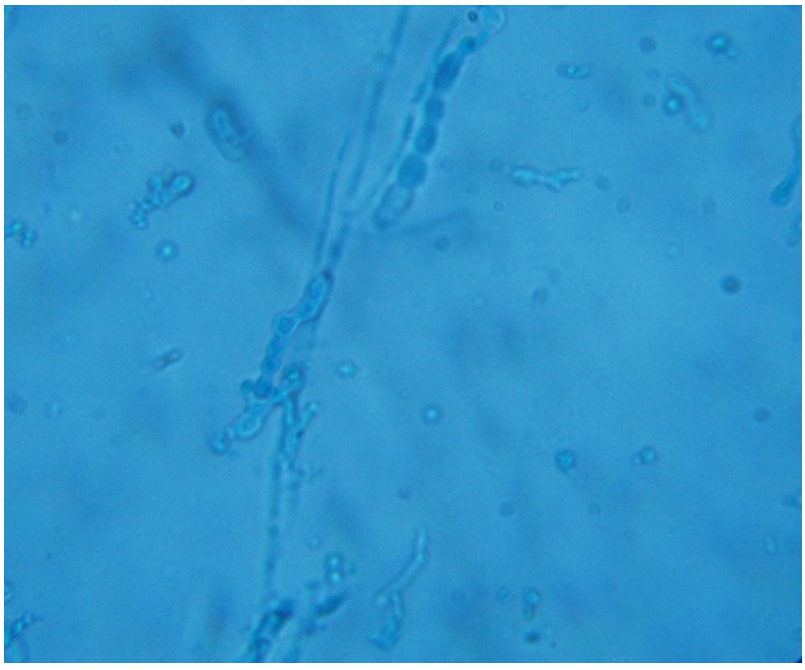


FIGURE 23 : MICROSCOPIC MORPHOLOGY – CHLAMYDOCONIDIA

TRICHOPHYTON SIMII

FIGURE 24: BUFF COLOURED COLONY WITH YELLOW REVERSE

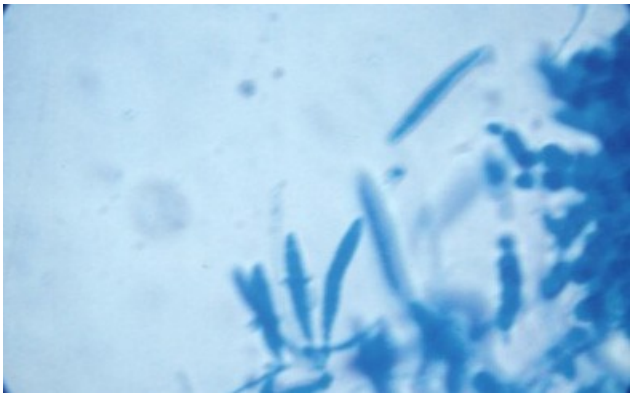


FIGURE 25: ENDOCHLAMYDOCONIDIA

TRICHOPHYTON VERRUCOSUM

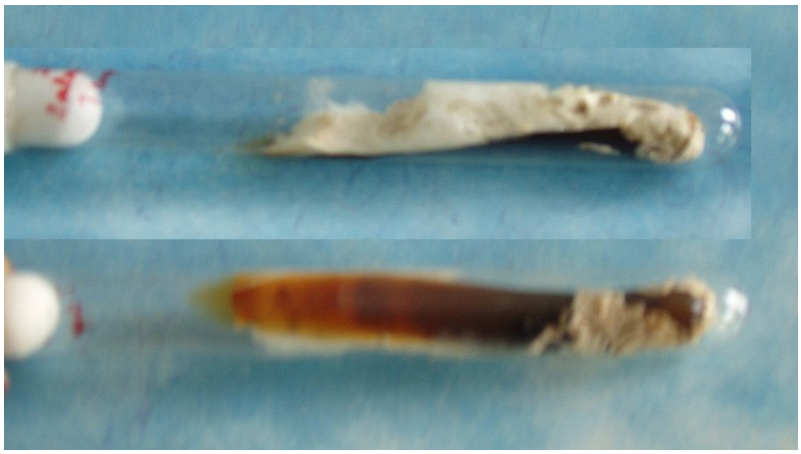


FIGURE 26: GRAY COLOURED COLONIES

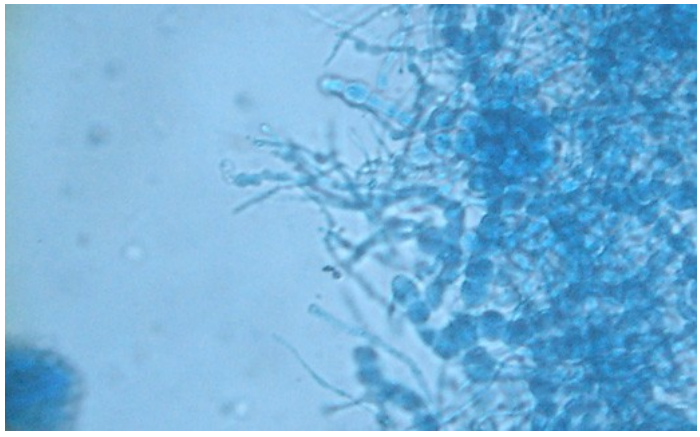


FIGURE 27: CHAINS OF CHLAMYDOCONIDIA

Blood group analysis was done in all the 60 patients (Table.5). Blood group O+ was found in majority (40%) of the

patients followed by B+ group (36%). A+ group was found in only 13% of the patients.

TABLE – 5 BLOOD GROUP ASSOCIATION

Blood group	No. of Patients	
	Rh+	Rh -
O	12	1
A	4	-
B	14	1
AB	3	-

DISCUSSION

In this study, the most affected age group was 30 to 40 years and the mean age was 47.8 years. Males were more affected in this age group. But most of the females were of 50 to 60 years age. This finding is consistent with that of a previous study conducted in our department where males were mostly affected in third decade and females in fourth to fifth decade of life. Their mean incidence of age was 39 years (Senthamil Selvi 1997)[30].

Females outnumbered males in the present study. The male : female ratio was 1 : 1.5. This is in contrast to earlier studies where males were predominantly affected (Senthamil Selvi 1997)[30], (Agarwalla A 2001)[43] (Sverjgard E, 1986)[36]. But female preponderance has been noted occasionally (Macura 1984).

Diabetes mellitus was the most frequent systemic association noted in this study. This is similar to the findings of some of the earlier studies (Senthamil Selvi 1995)[118] (Jolly and Carpenter 1968)[92]. But in certain studies of diabetes mellitus, neither increased prevalence of dermatophytsis nor any correlation between dermatophytosis and duration of diabetes have been noted (Buxton P.K. 1996)[91] (Romanio C. 2001) [115] (Lugo S.A. 1992)[40].

Bronchial asthma was the next common disease associated with chronic dermatophytosis in this study. Similar observation was made

in atopics with chronic dermatophytosis infection (Senthamil Selvi 1997)[30] (Hay RJ 1982)[28]. It has been found in certain studies that absorption of fungal allergen leads to bronchial hyperactivity and late onset intrinsic type asthma (Wood Folk JA 2005) (Hurliman A 2001)[88]. Atopy also can be exacerbated by chronic dermatophytosis because of the trichophytin hypersensitivity which is known as atopic – chronic – dermatophytosis syndrome. (Jones 1973)

Few cases of bronchial asthma were found to be associated with hypothyroidism and diabetes mellitus.

Most of these bronchial asthma patients were on long term systemic steroids. In many instances of chronic infection, long term steroids have been an associated factor (Rebell. G 1996).

Immunosuppressed states like HIV infection, Systemic lupus erythematosus, renal transplantation were noted in this study. Few cases of treatment failure were also observed. All these factors contribute to chronicity as noted in previous studies (Richard A.J. 2000)[48] (Wade K.F.2004)[2].

Tinea corporis and tinea glutealis were the frequently observed clinical types in this study. Waist in women, and back in men the commonest site affected as noted in previous studies. Tinea cruris was common in males while tinea axillaris was more in females. This type of presentation can be attributed to the dressing pattern of the patients

in this region. Occlusive dressing especially with synthetic clothes have always been considered as an important contributory factor due to the excessive sweating and moisture resulting in high humidity and CO_2 level (Micheal G. 2000)[53]. In tinea cruris, hypersensitivity to trichophytin antigen plays a role in pathogenicity (Chakravarthi 1995)[72].

Tinea corporis was the commonest presentation in diabetes mellitus patients.

About 78% of the study cases had multiple site involvement. Tinea unguium was noted in some of them. This could be a cause for the chronic multiple site infection as noted in certain studies[4].

Distal lateral subungual type of Tinea unguium was noted in most of the patients. One of the HIV infected patients had proximal subungual white onychomycosis. This is the most common type of nail tinea unguium seen in HIV infection[48].

Recurrent tinea unguium is usually found to be due to early termination of treatment, poor penetration of antifungals into the nails and underlying concomitant diseases.

One patient with bronchial asthma on long term steroids use had erythroderma like tinea corporis. Similar widespread, non inflammatory, resistant type of infections is usually noted in

immunosupressed states like HIV infection[48] and renal transplant patients [51,47,4].

Trichophyton rubrum was the most frequent isolate in this study. It was the causative of the erythroderma like tinea corporis observed in this study. Trichophyton rubrum usually causes widespread non inflammatory states [51,48]. This organism has been the commonest isolate in most of the previous studies (Rippon JW 1985)[63], (Senthamil Selvi, 1997)[30] (Wade KF, 2004)[2].

Trichophyton mentagrophytes was the second commonest isolate as observed in earlier studies[41] (Rippon JW 1988).

Trichophyton verrucosum was isolated from a HIV patient from rural area. It usually causes inflammatory type of dermatophytosis. But in this patient extensive non-inflammatory type of dermatophytosis was observed. Similar observation has been made previously in one study (Rippon JW 1985)[63].

Eosinophilia with absolute eosinophil count more than 450 cells mm^3 was noted in 56% of the study patients. Most of them had Diabetes and bronchial asthma. Eosinophilia along with elevated IgE has been reported to be associated with chronic dermatophytosis. (Martin E.S. 2003)[60] (Ward GW 1989)[89].

Blood group analysis showed ‘O’ to be the major group in this study. This finding is similar to that observed in a previous study

done at our centre (Senthamil Selvi 1995)[31]. No association between 'A' group and chronic dermatophytosis has been found in this study unlike previous studies (Neering H 1979)[109](Neilser P.G).[13](Romaro C2001) [76].

This shows that the ABO group in chronic dermatophytosis is consistent with the pattern seen in general population[110], 'O' group being the commonest type world wide[106].

CONCLUSION

- The 30 – 40 years age group was commonly affected by chronic dermatophytosis. The mean age was 47.8 years.

- Females were more affected than males.

- Diabetes mellitus and Bronchial asthma were the commonest associations. Other systemic conditions like hypothyroidism, systemic lupus erythematosus, chronic obstructive pulmonary disease, HIV infection, renal transplantation, hypertension and cutaneus disorder like ichthyosis vulgaris, palmoplantar psoariasis, discoid lupus erythematosus, keratolysis punctata, pemphigus vulgaris on steroids, angioedema and candidiasis were also observed.

- Tinea corporis was the commonest clinical pattern, especially in waist region in females and back in males. Tinea axillaris and tinea cruris were frequently noted in females and males respectively.

- *Trichophyton rubrum* was the commonest isolate with *Trichophyton mentagrophytes* being the second commonest one.

- Eosinophilia was observed in 56.6% of the patients. Most of them had diabetes mellitus and bronchial asthma.

- The frequently associated blood group was ‘O’. This is consistent with the pattern seen in general population.

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PROFORMA

Case No :

Name : Age : Sex :

Address : Occupation :

Diagnosis : Duration :

Contact History : Man -

Animal -

Family History :

Treatment History :

Other Drug Intake :

Clinical Examination :

Type of dermatophytosis		Surface Area (%)
Tinea corporis	:	
Tinea axillaris	:	
Tinea cruris	:	
Tinea glutealis	:	
Tinea genitalis	:	
Tinea manuum	:	
Tinea pedis	:	
Tinea unguium	:	
Tinea capitis	:	

Character Inflammatory / Non Inflammatory

Associated disorders

Skin Systemic

Investigations

1. Blood haemogram
2. Absolute eosinophil count
3. Blood urea and sugar
4. Blood group analysis
5. Potassium hydroxide mount
6. Culture

MASTER CHART

S. No	Age (yrs)	Sex	Clinical types	Duration of disease (yrs)	Surface area (%)	Associated diseases	Blood group	Absolute eosinophil count	Isolate
1.	45	F	T.glutealis T.cruris T.corporis T.axillaris	6	45	Diabetes mellitus. Hypertension.	B-	1100	T.rubrum
2.	39	F	T unguium	3	-	Plamoplantar Psoriasis Hypothyroidism	O+	960	T violaceum
3.	50	F	T.corporis T.cruris T.axillaris	20	9	Diabetes mellitus	B+	440	No Growth
4.	50	F	T.faciei T.corporis	6	18	Bronchial asthma	B+	400	T.rubrum
5.	23	M	T.corporis T.glutealis T.cruris	4	30	Irregular treatment	AB+	819	T.rubrum
6.	37	M	T.incognito	3	60	Bronchial asthma Systemic steroids	O+	520	T.mentagrophytes
7.	50	F	T.corporis T.unguium T.glutealis	6	40	Bronchial asthma	O+	580	-
8.	35	F	T.glutealis	3	10	Diabetes mellitus	B+	612	T.tonsurans
9.	28	M	T.cruris T.glutealis T.manuum	3	40	Oral candidiasis. HIV, contact with animals.	B+	525	T.verrucosum
10.	37	F	T.glutealis T.cruris T.corporis	3	30	Nil	O+	560	No growth
11.	50	F	T.corporis T.capitis	3	20	-	B+	490	No growth
12.	75	M	T.corporis T.cruris T.glutealis	3	40	Bronchial asthma Systemic steroids	B+	360	T.mentagrophytes
13.	47	M	T.corporis T.glutealis T.cruris	4	30		AB+	244	No growth
14.	62	M	T.cruris T.glutealis	3	40	COPD	A+	218	T.rubrum
15.	56	F	T.corporis T.glutealis	4	50	-	A+	747	No growth
16.	31	M	T.corporis T.ungruium	5	30	-	O+	280	T.rubrum
17.	45	F	T.manurum T.unguium	5	10	-	A+	570	No growth
18.	70	F	T.faciei	5	10	-	B+	630	No growth
19.	58	F	T.corporis T.glutealis	10	30	-	O+	490	T.mentagrophytes

20.	67	F	T.corporis T.glutealis	6	50	Bronchial Asthma	O+	580	-

S. NO	Age (yrs)	Sex	Clinical types	Duration of disease (yrs)	Surface area (%)	Associated diseases	Blood group	Absolute eosinophil count	Isolate
21.	54	F	T.corporis T.glutealis T.cruris	20	30	Diabetes mellitus with trophic ulcer	B+	480	No growth
22.	34	F	T.corporis T.unquium T.cruris	3	20	Diabetes mellitus	O+	480	-
23.	50	F	T.cruris T.corporis T.glutealis	5	30		B+	616	T.mentagrophytes
24.	30	M	T.corporis	3	10	HIV	B+	480	T.rubrum
25.	40	M	T.corporis	18	20		A+	1048	-
26.	44	M	T.corporis T.unguium T.cruris T.axillaris	4	30	Diabetes mellitus	B+	219	No Growth
27.	35	M	T.axillaris T.glutealis T.corporis	3	30		O+	310	No Growth
28.	54	M	T.corporis T.glutealis T.axillaris	10	40	Diabetes mellitus	O+	574	No Growth
29.	70	M	T.cruris T.corporis T.glutealis	3	30		B+	344	
30.	42	F	T.corporis T.cruris T.axillaris T.glutealis	10	30	Bronchial asthma Diabetes mellitus	A+	168	No growth
31.	40	M	T.corporis T.unguium T.glutealis	3	20	HIV	B+	264	T.rubrum
32.	55	F	T.corporis T.glutealis	4	20	Diabetes mellitus Hypertension	O+	1100	No growth
33.	35	F	T.glutealis T.corporis	20	20	Discoid lupsis erythematposis	B+	420	No growth
34.	60	F	T.corporis	5	20	Diabetes mellitus Hypertension	B+	560	T.rubrum
35.	69	M	T.cruris T.glutealis	10	10	Diabetes mellitus	O-	475	No growth
36.	36	M	T.corporis T.cruris	3	40	Contact with animals	O+	530	T.rubrum
37.	50	F	T.glutealis T.corporis T.axillaris T.unguium	6	40	Bronchial asthma, systemic steroids	B+	560	T.tonsuans
38.	55	F	T.corporis T.faciei	10	30		B+	630	No growth
39.	58	F	T.corporis T.glutealis	10	31	-	O+	490	T.mentagrophytes
40.	67	F	T.corporis T.glutealis	6	50	Bronchial asthma	O+	580	-

S. NO	Age (yrs)	Sex	Clinical types	Duration of disease (yrs)	Surface area (%)	Associated diseases	Blood group	Absolute eosinophil count	Isolate
41.	28	F	T.glutealis T.corporis	3	10	Angioedema	O+	400	No growth
42.	56	F	T.corporis T.glutealis	3	20	Diabetes mellitus Hypertension Coronary artery disease	O+	340	T.rubrum
43.	52	M	T.incognnito T.cruris T.glutealis	8	50	Topical steroids	O+	280	No growth
44.	37	M	T.glutealis T.corporis T.cruris	35	30	Hypertension	O+	540	-
45.	60	M	T.corporis T.cruris	3	20	Pemphigus vulgaris, diabetes mellitus, Steroids	O+	540	-
46.	39	M	T.corporis	20	20	Diabetesmellitus	A+	405	No Growth
47.	53	M	T.corporis	20	90	Bronchial asthma Systemic steroids	O+	440	T.rubrum
48.	42	F	T.corporis T.glutealis T.glutealis T.axillaris	3	40	Diabetes mellitus	O+	525	No Growth
49.	50	F	T.corporis T.cruris T.glutealis	5	30	Diabetes mellitus	B+	356	T.mentagrophytes
50.	50	F	T.corporis T.axillaris T.glutealis	10	50	Bronchial Asthma Hypothyroidism	B+	720	No growth
51.	32	F	T.corporis	3	10	NIL	AB+	700	T.simii
52.	32	M	T.unguim	20	-	Icthyosis vulgaris	B+	150	No growth
53.	32	F	T.corporis	3	20	HIV	O+	486	No growth
54.	45	M	T.corporis T.unguium	3	30	Renal transplant	O+	320	T.rubrum
55.	42	F	T.corporis T.glutealis T.axillaris	3	40	-	O+	1014	T.mentagophytes
56.	43	F	T.glutealis T.corporis	3	20	Diabetes mellitus	B+	280	T.mentagophytes
57.	35	M	T.corporis T.unguium	20	40	Irregular treatment	O+	260	-
58.	50	F	T.corporis T.glutealis	3	30	Discoid lupus erythematosus	B+	420	T.mentagrophytes
59.	70	F	T.corporis	20	30	Diabetes mellitus	A+	420	-
60.	35	M	T.corporis	3	20	-	B+	380	-